Papillary muscle repositioning for repair of anterior leaflet prolapse caused by chordal elongation

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Objective: Anterior leaflet prolapse is still a challenge. Various techniques have been described, but very little is known of the long-term outcome. We describe the long-term results of papillary muscle repositioning, with up to 15 years' follow-up.

Methods: From 1989 through 2005, 120 patients with anterior leaflet prolapse (97 bileaflet and 23 isolated anterior leaflet) were treated with papillary muscle repositioning when chordae were elongated. All patients had severe mitral regurgitation. The mean left ventricular end-systolic diameter on echocardiography was 39.4 ± 5.2 mm. The predominant cause was degenerative: dystrophic disease in 62 and Barlow's disease in 43. Papillary muscle repositioning was carried out on the posterior papillary muscle in 92.5% and on the anterior papillary muscle in 31.7%. A ring annuloplasty was performed in 117 cases. Fifty-seven (47.5%) patients had a tricuspid annuloplasty.

Results: There were no in-hospital deaths or patients lost to follow-up. Mean follow-up was 6.3 ± 0.4 years (maximum, 15.6 years). Cumulative actuarial survival at 5, 10, and 15 years was 97.2%, 94.1%, and 81.4%, respectively. Two (1.7%) patients required reoperation at 1 and 5 years after repair. No significant risk factor was identified for late mortality or reoperation. At the latest assessment, 88 (73.3%) patients were asymptomatic. Echocardiography showed no or trivial mitral regurgitation in 89 (74.2%) patients, mild mitral regurgitation in 8 patients, and moderate mitral regurgitation in 9 patients.

Conclusions: Anterior leaflet prolapse caused by elongated chordae can always be addressed with papillary muscle repositioning. Results indicate that it is a safe and durable technique, providing good long-term results in the management of degenerative pathology of the anterior leaflet.

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Mitral valve repair is the gold standard to treat mitral regurgitation.[1,2] Irrespective of the lesion, Carpentier has well shown that mitral valve repair is durable and offers the best therapeutic option with regard to left ventricular recovery and quality of life.[3,4] Quadrangular resection is an established surgical technique to repair posterior leaflet (PL) prolapse.[1] Some data still support the concept that the repair of anterior leaflet (AL) prolapse is more challenging and does not provide long-term results as good as those seen with PL prolapse.[5] AL prolapse can be treated with different techniques, such as chordal shortening[1] in case of elongated chordae, chordal transfer[6] in case of chordal rupture, and chordal substitution[7] in both cases. The fact is that chordal shortening has progressively been abandoned, whereas chordal substitution is increasingly accepted.[8] Recently, good long-term results have been reported with chordal substitution for AL prolapse, but these results remain inferior than those for PL prolapse.[9] In our experience we repair the AL prolapse by using a reproducible and safe technique: papillary muscle repositioning (PMR).[10,11] The aim of this study was to describe this technique and its long-term results to propose it as a reproducible procedure for AL prolapse caused by chordal elongation.

Abbreviations and Acronyms

AL = anterior leaflet
CS = chordal shortening
NYHA = New York Heart Association
PL = posterior leaflet
PMR = papillary muscle repositioning

Patients and Methods

Population and Mitral Valve Characteristics

Among 550 consecutive mitral valve repairs between 1989 and 2005, we have analyzed 120 patients who received AL repair with PMR in an isolated fashion or in conjunction with PL repair. There were 87 male and 33 female patients, with a mean age of 59.1 ± 11.5 years. Twenty-two (18%) of the patients were in New York Heart Association (NYHA) functional class I, 28 (23%) were in NYHA class II, 55 (46%) were in NYHA class III, and 15 (13%) were in NYHA class IV. Thirty-two (26.7%) patients were in atrial fibrillation. The preoperative echocardiographic data are described in Table 1. Thirty-seven (30.8%) patients had significant tricuspid regurgitation. The predominant cause of mitral regurgitation was degenerative: Barlow's disease (n = 43) and dystrophic disease (n = 62). The other causes were healed endocarditis (n = 5), rheumatic (n = 5), ischemic (n = 4), and congenital (n = 1). Barlow's disease was considered in front of a myxomatous mitral valve with excess tissue and with thick chordae and leaflets. It does more often take place in younger patients than dystrophic disease, which shows thin leaflets and chordae without excess tissue in older patients. An associated procedure was carried out in 76 (63.3%) patients: tricuspid annuloplasty in 57 (47.5%), coronary bypass in 11 (9.2%), aortic valve replacement in 4 (3.3%), the maze procedure in 2 (1.7%), aortic valvular plasty in 1 (0.8%), and the Yacoub procedure in 1 (0.8%). Indications for surgical intervention were severity of mitral regurgitation and end-systolic diameter of the left ventricle, with or without functional signs.

Anatomic Considerations

Anterior papillary muscle usually has 2 components: anterior and posterior. In contrast, posterior papillary muscle usually has 3 components: anterior, intermediate, and posterior (Figure 1). Chordae arising from the anterior head are anchored to the AL, chordae arising from the intermediate head are anchored to the commissural area, and chordae arising from the posterior head are anchored to the PL. Moreover, the anterior head is always higher than the posterior head. The splitting of the anterior head from the intermediate head and from the ventricular wall allows mobilization in any direction, especially downward into the ventricular cavity, as much as needed. Therefore all elongated chordae arising from the anterior head can be repositioned with the papillary muscle head being positioned downward, thus correcting the prolapse. With regard to papillary muscle vascularization, the split has to be vertical.

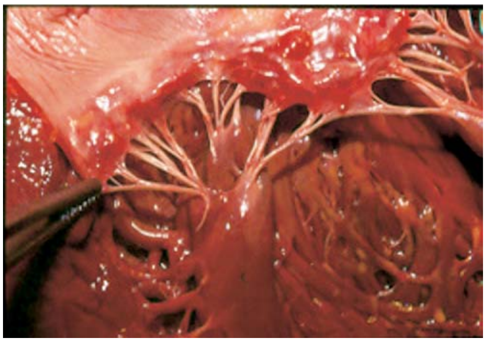

Figure 1. Posterior papillary muscle anatomy. Three heads can be identified: anterior, intermediate, and posterior. Chordae arising from the anterior head are anchored to the anterior leaflet, chordae arising from the intermediate head are anchored to the commissural area, and chordae arising from the posterior head are anchored to the posterior leaflet.

Operative Technique

Operations were performed during normothermia, and myocardial protection was achieved with cold crystalloid cardioplegia until 1993 and cold blood cardioplegia thereafter. AL prolapse was measured by means of transthoracic echocardiography preoperatively and defined by overriding the posterior mitral annulus by more than 5 mm. It was then confirmed by means of transoesophageal echocardiography intraoperatively in the longitudinal axis at the level of A2 P2. Finally, it was analyzed by the surgeon intraoperatively, as described by Alain Carpentier, when comparing all segments of the mitral valve with the reference point, most often with P1. Table 2 summarizes the surgical lesions of the mitral valve observed during the systematic intraoperative assess-

TABLE 1. Preoperative echocardiographic data

	Mean ± standard deviation
MR (grade 1/4 to 4/4)	3.6 ± 0.4
LVESD (mm)	39.4 ± 5.2
LVEDD (mm)	62.8 ± 4.8
LA size (mm)	49.4 ± 11
Systolic PAP (mm Hg)	47.9 ± 12.9
LVEF (%)	65.7 ± 8.9

MR, Mitral regurgitation; *LVESD*, left ventricular end-systolic diameter; *LVEDD*, left ventricular end-diastolic diameter; *LA*, left atrium; *PAP*, pulmonary artery pressure; *LVEF*, left ventricular ejection fraction.

TABLE 2. Intraoperative observed lesions

Specific lesion	N
Anterior leaflet prolapse	120
Posterior leaflet prolapse	97
Anterior commissural prolapse	3
Posterior commissural prolapse	16
Chordal rupture	53
Posterior leaflet restriction	7
Annular dilatation	112
Annular calcification	18

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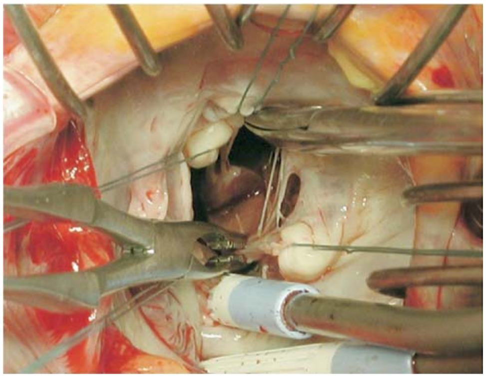

Figure 2. Surgical technique of posterior papillary muscle repositioning: identification and division of the heads of the posterior papillary muscle.

ment of all the segments. In all patients a posterior jet lesion was present along the PL and the posterior wall of the left atrium. When the prolapse involved the posterior part of the AL (A2/A3), we performed posterior PMR, and when it involved the anterior part (A1/A2), we performed anterior PMR. Great care was taken in understanding the chordal distribution to select the appropriate chordae. Associated repair techniques of the mitral valve were as described by Carpentier.[1]

First, the posterior PMR was performed as follows. As a first step, the anterior head was divided extensively from the intermediate head. In some instances some attachments with the left ventricular wall were also resected (Figure 2). A U stitch was then placed at the upper extremity of the anterior head into its fibrous part. The appropriate shortening was then assessed by pulling the anterior head downward. The degree of the prolapse was assessed accurately by using 2 hooks, one pulling the free edge of the prolapsed AL and the other pulling the free edge of the PL adjacent to the AL. This maneuver gives the exact height of the prolapse. Consequently, the displacement of the papillary muscle equals the extent of the prolapse previously measured. The stitch previously attached to the anterior head was anchored secondarily to the fibrous tissue of the posterior head or even deeper into the muscular part of the posterior head if needed (Figure 3). In such instances the suture was pledgeted with a piece of autologous pericardium. The appropriate location was determined by the height of the prolapse (Figure 4).

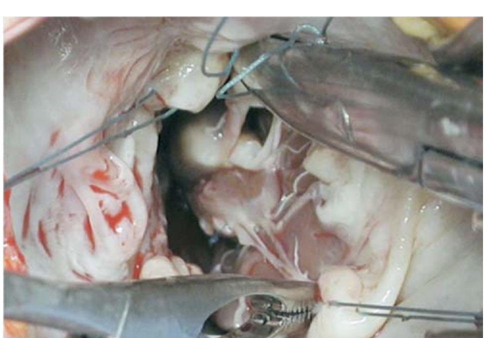

Figure 3. Surgical technique of posterior papillary muscle repositioning. A U stitch is made in the fibrous segment of the anterior head and tied to the fibrous segment of the posterior head.

Second, anterior PMR was performed as follows. The anterior head of the anterior papillary muscle encompassing the chordae going to A1/A2 was identified and split from the posterior head. Then a U stitch was placed at the upper extremity of the anterior head into its fibrous part, which was then anchored lower to the posterior head of the anterior papillary muscle, therefore bringing down the free edge of A1/A2 by appropriate length lower into the ventricle.

All but 3 patients received a Carpentier-Edwards rigid prosthetic ring. The measurement for the adequate ring size was performed in a conventional manner. The surface of the ring was at least equal to the surface of the AL.[1] The distance between the anterior and posterior commissures was also respected. The fibrous trigone was not plicated. The mean size of the mitral ring was 32.4 ± 1.5 mm. The majority of the rings were size 32, and no patient received a ring less than 28 mm in size. A combination of several techniques was necessary to achieve the repair in such instances. Table 3 summarizes all the surgical techniques used in this series of patients. Patients with coronary artery disease had distal, as well as proximal, anastomoses performed after the valve repair. If an aortic or tricuspid valve correction was needed, it was performed after the mitral valve repair. Tricuspid annuloplasty was performed only if the tricuspid annular diameter was greater than twice the normal size (≥70 mm), regardless of the grade of regurgitation.[12] The mean of the tricuspid ring

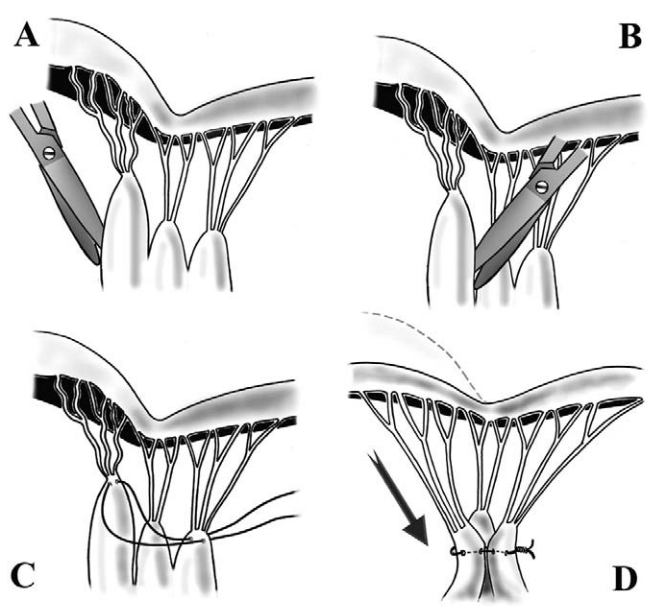

Figure 4. Schematic drawing of the technique. A, Resection of attachments between the ventricular wall and the posterior head of the papillary muscle. B, The posterior head is split from the intermediate head. C, A U stitch is placed in both the anterior head and the posterior head. D, The anterior head is brought down, fixing the anterior prolapse.

TABLE 3. Surgical procedures

Anterior leaflet		Posterior leaflet	
PPMR	111	Quadrangular resection	85
APMR	38	Sliding plasty	59
Leaflet resection	17	Posterior leaflet preservation	8
Posterior chordal shortening	4	Chordal substitution	6
Anterior chordal shortening	35	Leaflet mobilization	1
Chordal transfer	13	Leaflet patching	2
Chordal substitution	3	Annular decalcification	16
Commissural sliding	14	Ring annuloplasty	117
Pericardial patching	2		

PPMR, Posterior papillary muscle repositioning; *APMR*, anterior papillary muscle repositioning.

was 33.5 ± 0.8 mm. The mean aortic crossclamp time and cardiopulmonary bypass time were 98.2 ± 22.7 minutes and 124 ± 27.7 minutes, respectively.

Follow-up

Intraoperative transesophageal echocardiograms were obtained for all patients. Clinical and echocardiographic examinations were performed in all patients before hospital discharge, at 1 month, and then every year by the referring cardiologist. On echocardiography, mitral valve regurgitation was graded from 1 to 4. The mean follow-up time was 6.3 ± 0.4 years, and the median follow-up time was 5.9 years (range, 0.1-15.6 years). Our study followed all the ethical rules of the United Kingdom.

Statistical Analysis

Description of continuous variables was expressed as the mean ± standard deviation of the mean or as the median with the extremes. Categoric variables were presented as absolute numbers of patients and percentages. The statistical significance of the comparisons between 2 or several groups was tested by using a log-rank test. Survival was calculated with the Kaplan-Meier method. The analyses were carried out with SPSS software (version 11.5.1, SPSS Inc).

Results

Early Results

Postoperative mortality and morbidity. There were no in-hospital deaths. Eight (6.5%) patients required an early operation: 7 pericardial drainages for hemopericardium and 1 for mediastinitis. There was 1 transient cerebral ischemic attack. There was no systolic anterior motion of the anterior mitral leaflet, no hemolysis, and no infective endocarditis. There was no early mitral valve failure and no reoperation for it.

Postoperative echocardiographic results. Transthoracic echocardiography at discharge showed no residual mitral insufficiency in 98 (81.7%) patients, trivial regurgitation in 10 (8.3%) patients, and mild regurgitation in 12 (10%) patients. The mean of mitral regurgitation was 0.16 ± 0.16, and the mean of the diastolic pressure gradient across the mitral valve was 3.39 ± 1.15 mm Hg.

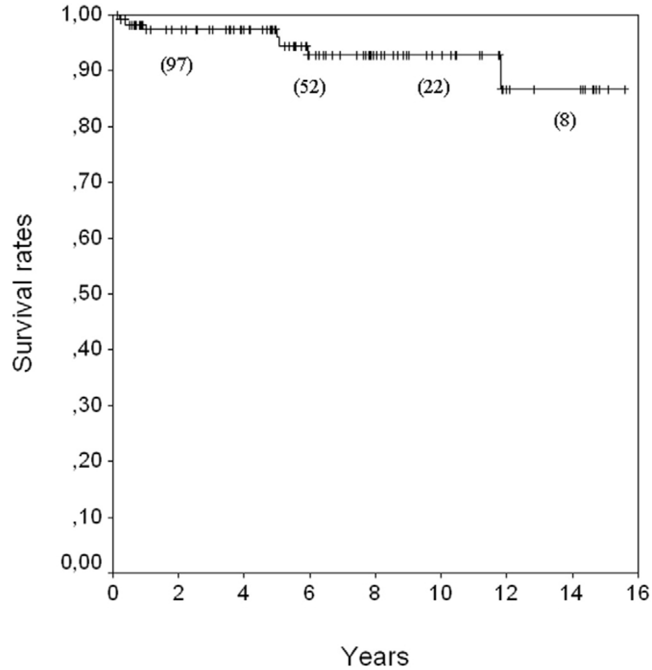

Figure 5. Actuarial survival curve including only cardiac deaths. Numbers in parentheses are subjects at risk.

Postoperative conduction and rhythm. Ten (8.3%) patients were in complete atrioventricular block, requiring a permanent pacing. Eighty (66.5%) patients were in sinus rhythm, 30 (25%) were in atrial fibrillation, 4 (3.5%) were in junctional rhythm, and 6 (5%) were in ventricular rhythm.

Late Results

Eighty-eight (73.3%) patients were in NYHA class I, 15 were in NYHA class II (12.5%), and 3 were in NYHA class III (2.5%). At follow-up transthoracic echocardiography, no insufficiency or a minimal insufficiency was present in 89 (74.2%) patients, mild insufficiency was present in 8 (6.7%) patients, and moderate insufficiency was present in 9 (7.5%) patients. There were 14 (11.7%) late deaths, including 7 (5.8%) of cardiac causes. Considering only these deaths from cardiac causes, the cumulative actuarial survival rates at 1, 5, 10, and 15 years were 98.3%, 97.2%, 94.1%, and 81.4%, respectively (Figure 5). The causes of death linked with the heart were heart failure (n = 1), pulmonary embolism (n = 1), pulmonary edema (n = 1), and cerebral hemorrhage after mitral valve replacement (n = 1). The other causes of deaths were stroke (n = 4), sepsis caused by diabetes (n = 1), mesenteric infarction (n = 1), and pneu-

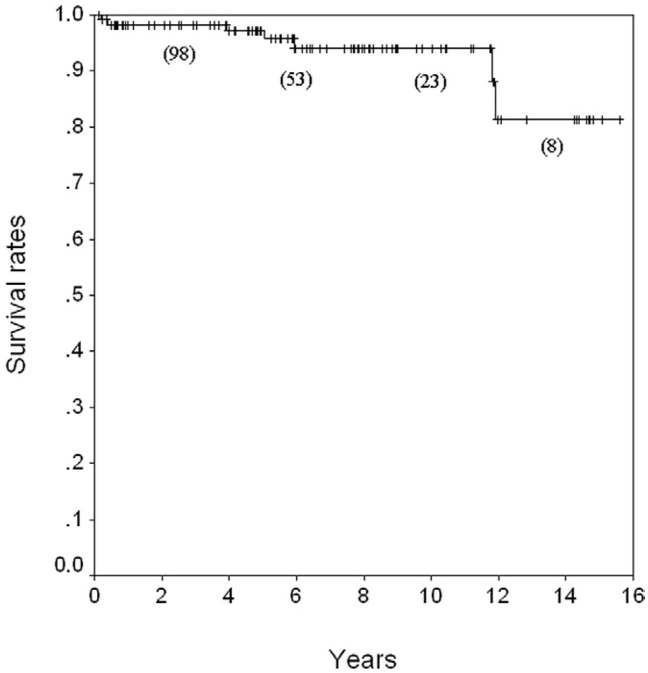

Figure 6. Freedom from reoperation curve. Numbers in parentheses are subjects at risk.

monia (n = 1). A reoperation was required in 2 (1.7%) patients, who have had mitral valve replacement for severe mitral regurgitation caused by failure of the repair 1 and 5 years after the repair, respectively. These 2 patients died 3 and 6 years after the replacement, respectively. The cumulative actuarial survival rates for freedom from reoperation involving the mitral valve at 1, 5, 10, and 15 years were 97.4%, 97.4%, 92.8%, and 86.7%, respectively (Figure 6). Age (P = .14), sex (P = .43), diabetes (P = .63), chronic renal insufficiency (P = .59), cause (dystrophic or Barlow's disease, P = .45), associated PL prolapse (P = .10), previous rhythm (P = .06), and concomitant surgical procedure (P = .87) were not statistically predictive factors for death. Associated PL prolapse (P = .51), posterior commissural prolapse (P = .66), anterior commissural prolapse (P = .92), annular decalcification (P = .59), and chordal substitution (P = .79) were not statistically predictive factors for reoperation.

Discussion

Excellent results have been obtained with mitral valve repair, avoiding the inconveniences of prosthetic replacement. In fact, mitral valve repair is durable and presents a very low rate of valve-related events[13-16] and should be the procedure of choice to treat degenerative mitral insufficiency. PL repair is the most common procedure in mitral valve repair.[1,3,4,17] Quadrangular resection with or without a sliding plasty is the gold standard in this condition. Prosthetic ring annuloplasty to reshape the mitral orifice in all chronic diseases in the above cases is accepted by the majority of the surgeons.[11] The policy is to implant the biggest ring possible because ring insertion is not used to restrict the mitral valve surface but only to reshape the orifice.[11]

However, there might still be some concerns about the AL prolapse, which can remain a surgical challenge. There is no consensus as to how AL prolapse should be repaired, and some have even raised the issue of whether to neglect AL prolapse.[18] It seems crucial to emphasize that billowing AL and AL prolapse are not identical. Billowing can be seen in excess of 70% of the population without any regurgitation, and of course billowing does not require any corrective measure. On the contrary, regurgitation cannot take place without prolapse. It seems true that 2 mm of AL prolapse can be addressed by using ring annuloplasty alone because it decreases the aortomitral angle and pulls the free edge of the AL downward. However, AL prolapse in our series was always more than 5 mm and therefore received a specific treatment. Combination of PL prolapse with Al prolapse takes place in 20% to 30% of cases of degenerative mitral valve disease, and we have outlined the difference between billowing and prolapse, although this does not mean that we have corrected prolapse that did not require surgical treatment. Moreover, this concept that AL combined with PL does not require specific treatment can account for some of the poor results in series previously published by others. We believe that AL repair can be as easy and reliable as PL repair. The first step is to have a good knowledge of the mitral subvalvular apparatus, especially the papillary muscles and their heads. In our series isolated PMR became the single and easy technique to repair AL prolapse in all locations of the lesions (ie, A1, A2, and A3) and was provided as first-intention treatment. PMR can only treat chordal elongation. When the anterior head is not really individualized, it is possible to create it by creating a trench into the papillary muscle between the anterior and the posterior chordae. It is important to stress that the shortening should always be a bit less than needed to avoid restricted motion of the AL. In such instances combined techniques were necessary to achieve a complete correction of the regurgitation. Limitations can be seen when elongated chordae are too long. In such instances we have completed the repair by using chordal plication at the level of leaflet attachment. In case of mixed lesions, such as chordal elongation and chordal rupture, we have associated PMR with chordal transfer.

However, we believe that there are many reasons to identify PMR as the method of choice:

- Chordal transfer (PL to AL) is an excellent method but can be limited by the number of chordae available to treat extensive AL leaflet prolapse.
- Chordal shortening (CS) is not a direct method to shorten elongated chordae because the effective short-

ening represents half of the length buried into the trench of the papillary muscle.[1] Consequently, CS requires experience in the field of mitral valve repair. On the contrary, PMR is a direct shortening because the repositioning deeper into the left ventricular cavity equals the length of the prolapse. Therefore we believe that this technique is easier and safer.

- Moreover, CS requires great care to avoid the buried sutures to be in contact with the shortened chordae because it can induce rupture with time. Probably because of this technical aspect, some researchers, such as Gillinov and colleagues,[19] reported a failure rate of 22% in their valve repair when using CS, which increased up to 36% in degenerative disease.[20] Although we did not find this complication in our personal experience with more than 400 mitral valve repairs, such a failure rate might explain the need to search for an alternative technique.
- PMR can be mostly useful in case of paramedian and paracommissural posterior prolapse of the AL. In many patients the chordae usually arise from the tip of the posterior papillary muscle. Therefore chordal burying becomes impossible because rarely more than 2 chordae can be buried into the same trench. In those instances PMR offers a safe and elegant alternative option. The basic principle of this technique is to split the anterior head of the posterior papillary muscle to pull a few chordae separately deep down into the ventricle, independent of the others.
- When paramedian AL prolapse occurs, very often the posterior commissural area is not involved. In such cases the splitting of the anterior head corrects the localized prolapse without interfering with the adjacent structures of the leaflet.
- In most instances repositioning requires only one 4-0 monofilament suture tied into the fibrous area of the head of the papillary muscle. This is the simplest and fastest technique to correct AL prolapse. We have always been able to perform it.
- When AL prolapse occurs at the level of the anterior papillary muscle, PMR is also feasible. In most instances there are only 2 components, anterior and posterior, that can be easily split, and then the same technique can be used. Moreover, posterior PMR can be easily associated with anterior PMR. Therefore any prolapsed area of the AL can be successfully treated with this technique.
- Posterior commissural prolapse might still remain the most challenging lesion to repair. In our series we have found 16 (13.3%) patients showing such a lesion associated with an AL prolapse. By separating the anterior and the intermediate head from the posterior head, it allows us to shorten, to a different extent, the commissural chordae from the paramedian chordae. Recently, Aubert and associates[21] described long-term outcome for commissural prolapse repair with good results, but in 50% of the cases, they closed the commissure. In our series we never closed the commissure, and it was always effectively done with PMR of the intermediate head of the posterior papillary muscle.

We strongly believe that PMR shows better results than the other methods used previously because no reoperation for recurrent AL prolapse was required in our previous study at 10 years.[10] Mohty and coworkers[5] have shown in a very elegant study that between the 1980s and 1990s, the risk rate for reoperation of AL repair had gone down from 25% to 10%. There was still a difference in the outcome between PL and AL. Our study shows that it is possible to avoid this difference and decrease the reoperation rate for recurrent mitral regurgitation. We believe that PMR does not require an extensive learning curve, and because it is a fast technique, it saves time for other maneuvers to completely treat AL prolapse. David and colleagues[13] have stated that "prolapse of multiple segments of both leaflets may be better served by valve replacement than by valve repair until newer reconstruction approaches, such as shortening of the entire papillary muscle trunk are proved satisfactory." Although we do not propose to displace the entire papillary muscle, we believe that partial papillary muscle displacement can provide a safe, durable, and reproducible technique to correct the most complex lesions of the AL, such as those seen in Barlow's disease, with or without posterior commissural prolapse and extensive valvular tissue.

Conclusions

In our experience PMR is the method of choice to treat AL prolapse, irrespective of the location of the lesion, with excellent clinical and echocardiographic long-term results. We recommend this method for all AL chordal elongation and especially for commissural prolapse. It provides good long-term results that are as favorable as those obtained with repair of PL prolapse.

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